

Lifetime episodes of dysphoria: Gender, early childhood loss and personality

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The roles of gender, early childhood loss and personality as risk factors for lifetime episodes of dysphoria were examined in a large sample of college students ($N = 557$). Dysphoria classifications were based on the Inventory to Diagnose Depression (IDD) and the IDD-Lifetime Version. Brief dysphoria was defined as meeting DSM-III-R symptom criteria for major depression without meeting the two-week duration criteria, whereas protracted dysphoria required that the duration criteria were also met. Although females reported higher levels of depressive symptoms and neuroticism than did males, and were more likely to have a lifetime history of episodes of dysphoria, males were more susceptible to the adverse effects of early childhood loss. Males with loss were more likely to have a history of protracted dysphoria and to report higher levels of neuroticism than were males who did not experience an early parental loss. Neuroticism, in turn, acted as a trait vulnerability characteristic to episodes of dysphoria in both males and females. Interestingly, the effects of gender on lifetime experience of dysphoria were mediated by neuroticism: females' increased vulnerability to episodes of dysphoria was due to their elevated levels of neuroticism.

Although researchers and theoreticians have a long-standing interest in psycho-social vulnerability to various forms of emotional distress, such as depression, there is still little agreement concerning the roles of such factors. The current study focuses on three putative risk factors to lifetime episodes of dysphoria: gender; early childhood loss; and the personality dimensions of neuroticism and extraversion-introversion. Due to controversy concerning the validity with which major depressive disorder can be diagnosed with self-report measures (Coyne, 1994), in the present study we label self-reported conditions that meet full symptom criteria for major depression, as defined by the *Diagnostic and Statistical Manual, Third Edition-Revised* (DSM-III-R; American Psychiatric Association, 1987), as episodes of dysphoria.

One of the most consistent findings in the area of depression is that females are at greater risk than males for developing the disorder (Nolen-Hoeksema, 1987). Women have a greater lifetime prevalence of major depressive disorder (Blazer, Kessler, McGonagle & Swartz, 1994) and a higher point prevalence of depression

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(Blazer *et al.*, 1994; Regier *et al.*, 1984; Weissman & Klerman, 1977). Further, in community samples women generally report greater severity of depressive symptoms than do men (Eaton & Kessler, 1981; Ferichs, Aneshensel & Clark, 1981), although this gender difference is found less consistently in college samples (e.g. Hammen & Padesky, 1977). Nevertheless, it is unclear what accounts for higher levels of depression in women. One possibility is that women have more, or a higher level, of the characteristics that place both genders at risk for developing depression. For example, Nolen-Hoeksema (1987, 1991) suggested that women are more likely to respond to depressed mood states and symptoms with rumination. Rumination, in turn, is thought to lead to more persistent and severe episodes of depressive mood and symptomatology (e.g. Morrow & Nolen-Hoeksema, 1990). In a recent study, Nolen-Hoeksema, Parker & Larson (1994) provided support for this mediational model: gender had only an indirect effect on future depressive symptoms through its impact on rumination. From a somewhat different perspective, women might be more likely to have other personality features and characteristics that are believed to confer vulnerability to depression. As we discuss below, neuroticism is one such plausible candidate. Finally, it also is possible that women are more *sensitive* than are men to the depressogenic effects of psychological characteristics, such as neuroticism, or adverse experiences, such as parental divorce during childhood (see Rodgers, 1994).

In contrast to gender differences, there is less consistent evidence that loss during childhood, through parental death or divorce, contributes to depression in adulthood (Parker, 1992). Whereas some studies have found that early parental loss is associated with subsequent depression (e.g. Brown & Harris, 1978; Kessler & Magee, 1993), other investigators have reported that his effect is small or non-existent (e.g. Kendler, Neale, Kessler, Heath, & Eaves, 1992; Tennant, 1988). The equivocal nature of these findings has led reviewers to suggest that more specific processes that occur following the loss, such as dysfunctional parenting styles and family conflict, are critical in developmental pathways leading to depression (Parker, 1992). According to this argument, psychological well-being is not so much dependent on early parental loss *per se* as it is on its after-effects in terms of family atmosphere and parenting. In the case of parental divorce, conflict between parents both before and after the divorce appears to account for much of the psychological sequelae (Emery, 1982; Franklin, Janoff-Bulman & Roberts, 1990; Gotlib & Avison, 1993). Similarly, quality of parenting appears to be a critical link in the causal chain between childhood loss and adult depression (Harris, Brown & Bifulco, 1990a, b).

Although these types of environmental mediators are likely to be important, there also is evidence that intra-psychic mediators, such as working models of attachment relationships, play a significant role in the development of depression (Carnelley, Pietromonaco & Jaffe, 1994; Roberts, Gotlib & Kassel, 1996b; Whisman & Garvey, 1995). Therefore, it is plausible that early childhood loss and adversity are associated with the development of personality features and characteristics that, in turn, mediate the relation between loss and depression. For example, the parental conflict and hostility that often accompany divorce might contribute to the development of specific personality styles in children that, in turn, are associated with risk for devel-

oping episodes of depression later in life (Kendler, Kessler, Neale, Heath & Eaves, 1993). Two such personality styles are neuroticism and introversion.

Neuroticism is defined as a proclivity to experience negative affective states and affective instability (Costa & McCrae, 1980; Eysenck & Eysenck, 1985). This personality dimension is characterized by a hypervigilant, ruminative and introspective cognitive style (Watson & Clark, 1984; Watson & Pennebaker, 1989) that negatively biases cognitive processes (Martin, 1985) and increases reactivity to stressful daily life-events (Bolger & Schilling, 1991). A number of studies have linked higher levels of neuroticism not only to current depression (e.g. Kendell & DiScipio, 1968; Matussek & Feil, 1983), but also to depression proneness. For example, Boyce, Parker, Barnett, Cooney & Smith (1991) found that high levels of neuroticism measured antenatally were associated with a threefold increase in risk for post-partum depression. Hirschfeld *et al.* (1989) found a similar prospective relation between neuroticism and the onset of major depression in a large sample of relatives of depressed patients. More recently, Kendler *et al.* (1993) demonstrated that neuroticism made a significant contribution to the prospective prediction of the onset of major depression that was independent of genetic risk. Finally, several investigators have found that higher levels of neuroticism are associated with a more pernicious course of depression (e.g. Duggan, Lee & Murray, 1990; see Clark, Watson & Mineka, 1994, for a review).

Although these studies have demonstrated prospective associations between neuroticism and the onset and course of depression, less research has examined whether previously depressed individuals are characterized by elevated levels of neuroticism. Interestingly, several studies have found that levels of neuroticism in recovered depressives are comparable to well-established norms for the general population (see Clark *et al.*, 1994). Such findings raise questions concerning the validity of the formulation that neuroticism is a trait vulnerability factor that is apparent before, during and after episodes of depression. It is important to realize, however, that such norms are based on general population samples which, given the relatively high base rates of depression, include many individuals with current and past episodes of the disorder. Consequently, normative values of neuroticism are likely higher than those found in samples of healthy, never depressed individuals (Clark *et al.*, 1994).

Finally, extraversion-introversion is another major dimension of personality (Costa & McCrae, 1980; Eysenck & Eysenck, 1985) that has been implicated in depression. For example, in their review of psychosocial factors in depression, Barnett & Gotlib (1988) identified introversion as an enduring abnormality in the functioning of remitted depressives. Consistent with these findings, recent theoretical and empirical work suggests that low positive affect differentiates depression from anxiety disorders, whereas high negative affect is common to both (Clark & Watson, 1991; Watson, Clark & Carey, 1988). Because extraversion is considered to be a personality dimension that reflects a proclivity to experience positive affect (Fowles, 1994; Larsen & Ketelaar, 1991), extraversion-introversion should be more strongly linked to depression than it is to anxiety. Nevertheless, in a recent review Clark *et al.* (1994) suggested that the empirical evidence is not clear regarding the role of introversion as a trait vulnerability factor in depression.

The purpose of the present study was to examine the roles of gender, early childhood loss and the personality dimensions of neuroticism and extraversion-introversion as risk factors for lifetime episodes of dysphoria in a large sample of college students. With respect to these factors, we hypothesized that females, individuals whose parents divorced or died before they were aged 11, and relatively more neurotic and introverted participants would be prone to lifetime episodes of dysphoria. We also tested whether gender interacted with these other putative risk factors in predicting lifetime episodes of dysphoria. Finally, we tested a mediational model that suggests that gender and early childhood loss increase an individual's risk for lifetime episodes of dysphoria through their association with neuroticism, which more directly confers vulnerability. In contrast to the practice of many previous studies, we felt that it was important to control for subclinical symptoms in analyses examining personality (cf. Barnett & Gotlib, 1988). Previously depressed persons tend to be more symptomatic than never depressed controls (Depue & Monroe, 1986), and such mild dysphoria could negatively bias personality ratings (Widiger, 1993). Thus, apparent differences in personality found in previous studies could be due to differences in subclinical symptomatology. Indeed, Maier, Lichtermann, Minges & Heun (1992) argued that neuroticism might reflect an attenuated form of depression.

Method

Participants and procedure

The initial sample of participants was composed of 694 undergraduates at Northwestern University who participated in this study as part of a course requirement for introductory psychology. An extensive battery of questionnaires was completed in mass testing sessions. Analyses were restricted to the 557 individuals (297 female) for whom there were complete data on the central variables, and who reported that they were neither adopted nor had experienced the loss of a parent between the ages of 11 and 18 years (see below). Although age and class year were not coded, the vast majority of introductory psychology students at Northwestern University are 18-year-old freshmen.

Measures

Inventory to Diagnose Depression. The Inventory to Diagnose Depression (IDD; Zimmerman, Coryell, Corenthal & Wilson, 1986) was used to measure severity of depressive symptoms, as well as to determine whether DSM-III-R criteria for major depressive disorder were met. Coefficient alpha was .92 in two independent samples (Zimmerman & Coryell, 1987; Zimmerman *et al.*, 1986), and a kappa coefficient of .8 was found between the IDD and the Diagnostic Interview Schedule (DIS) when these two measures were completed within two days of each other (Zimmerman & Coryell, 1988). Further, when treated as a continuous measure of severity, the IDD correlates highly with the Beck Depression Inventory and the Hamilton Rating Scale (Zimmerman *et al.*, 1986). The IDD-Lifetime (IDD-L; Zimmerman & Coryell, 1987) was used to assess participants' worst lifetime experience of depression. The IDD-L has good sensitivity (.74) and specificity (.93) when compared against the DIS, with a kappa coefficient of .6 (Zimmerman & Coryell, 1987).

In terms of lifetime experience of episodes of dysphoria, participants were classified as having: (a) brief current dysphoria (BCD) if they met DSM-III-R symptom criteria for major depression but not the two-week duration criteria; (b) protracted current dysphoria (PCD) if they met both DSM-III-R

symptom and duration criteria; (c) brief previous dysphoria (BPD) if they met DSM-III-R symptom, but not duration, criteria at some point in their lives and were not currently dysphoric; (d) protracted previous dysphoria (PPD) if they met DSM-III-R symptom and duration criteria at some point in their lives and were not currently dysphoric; and as (e) never dysphoric (ND) if they failed to meet DSM-III-R symptom criteria for major depression at any point in their lives.

Personality. The Eysenck Personality Inventory (EPI; Eysenck & Eysenck, 1964) was used to measure neuroticism and extraversion-introversion. Psychological defensiveness was measured using the Lie scale from this inventory. The EPI is a 57-item self-report inventory with good internal reliabilities (e.g. Horwood & Fergusson, 1986; Roberts, Gotlib & Kassel, 1996b).

Childhood loss. A stem question asked participants if they had been raised by both biological parents through the age of 18. If participants answered 'no' to the stem question, they were asked if a parent had died before the age of 11 and if their parents had divorced or permanently separated before the age of 11. Individuals who indicated that they had not been raised by both biological parents until the age of 18, but who had not experienced a parental death or divorce before age 11, were excluded from the analyses ($N = 29$). This group was composed of adoptees and of individuals with forms of loss occurring after the age of 11.

Results

Descriptive statistics

Eleven participants reported that, before they reached the age of 11 years, one of their parents died, and 67 participants reported that their parents divorced before this age. For the purpose of analyses, these two groups of participants were combined and are referred to as the parental loss group ($N = 78$). At the time of testing, 14 (2.5 per cent) individuals had PCD, 12 (2.2 per cent) had BCD, 87 (15.6 per cent) had PPD, 141 (25.3 per cent) had BPD, and 303 individuals (54.4 per cent) were never dysphoric during their lifetimes. Consistent with our selection procedure, the five groups of participants differed in terms of severity of current depressive symptoms ($F(4,552) = 125.74, p < .001$). Follow-up Tukey tests revealed that all groups differed from each other (all $ps < .001$), except for the PCD and BCD groups. As can be seen in Table 1, BCD and PCD participants reported the highest level of depressive symptomatology, followed by the PPD participants, then the BPD participants, and finally, ND persons.

As can be seen in Table 2, neuroticism was strongly correlated with both current ($r = .60$) and worst lifetime ($r = .50$) severity of depressive symptoms. In contrast, extraversion was only weakly correlated with these measures of depressive symptoms (all $rs < |.16|$). Defensiveness was not highly correlated with any of the symptom or personality measures (all $rs < |.19|$), suggesting that the associations among these variables were not due to social desirability.

Group comparisons

Gender. Consistent with the results of previous studies, there was a significant association between gender and symptoms of depression. Compared with males, females

Table 1. Mean depression and personality scores across dysphoria groups

Measures	BCD (N = 14)		PCD (N = 12)		BPD (N = 141)		PPD (N = 87)		ND (N = 303)	
	M	SD	M	SD	M	SD	M	SD	M	SD
Current IDD	32.5 _a	6.0	27.9 _a	3.9	12.0 _b	5.7	15.3 _c	6.7	8.0 _d	4.7
Neuroticism	17.8 _a	2.6	16.3 _{ab}	3.3	12.4 _c	4.4	13.2 _{bc}	3.6	9.3 _d	4.2
Extraversion- introversion	11.4 _a	5.8	11.9 _a	3.0	12.4 _a	3.9	12.3 _a	4.6	12.9 _a	4.0
Defensiveness	2.9 _a	1.4	2.1 _a	1.4	2.3 _a	1.5	2.3 _a	1.5	2.5 _a	1.6

Note. IDD = Inventory to Diagnose Depression; BCP = brief current dysphoria; PCD = protracted current dysphoria; BPD = brief previous dysphoria; PPD = protracted previous dysphoria; ND = never dysphoric. Means with similar subscripts are not statistically different at $p < .05$.

Table 2. Correlation matrix, means and standard deviations of continuous measures

	1	2	3	4	M	SD	N
1. IDD-Current					11.2	7.3	557
2. IDD-Lifetime	.59**				24.4	12.4	549
3. Neuroticism	.60**	.50**			11.1	4.7	557
4. Introversion-extraversion	-.15**	-.06	-.21**		12.6	4.1	557
5. Defensiveness	-.11*	-.12*	-.18**	-.18**	2.4	1.6	557

* $p < .01$; ** $p < .001$.

Note: IDD = Inventory to Diagnose Depression.

reported both more severe current depressive symptomatology (11.9 vs. 10.4; $t(555) = 2.38, p < .05$), and more severe worst lifetime depressions (27.0 vs. 21.5; $t(547) = 5.32, p < .001$). Similarly, and as can be seen in Table 3, females were more likely to have a lifetime history of episodes of dysphoria ($\chi^2(4, N = 557) = 12.27, p < .05$). Consistent with our mediational model, females also scored higher than males on neuroticism (12.1 vs. 9.8; $t(555) = 6.07, p < .001$). There were no gender differences with respect to either extraversion ($t(555) < 1$) or defensiveness ($t(555) = 1.17, p > .05$).

Childhood loss. As can be seen in Table 4, there was a non-significant trend for individuals who experienced parental loss before the age of 11 years to be more likely to have a history of dysphoric episodes than did those without loss ($\chi^2(4, N = 557) = 8.62, p < .08$). In particular, persons with loss had elevated rates of PPD compared with those without loss (25.6 per cent vs. 14.0 per cent). Interestingly, among males, early parental loss was significantly associated with an increased likelihood of having a lifetime history of dysphoric episodes ($\chi^2(4, N = 260) = 14.88, p < .01$); this effect was not significant among females ($\chi^2(4, N = 297) = 6.09, p > .10$). Whereas

Table 3. Proportions of males and females across dysphoria groups

	Males (<i>N</i> = 260) (%)	Females (<i>N</i> = 297) (%)
Brief current dysphoria	2.7	2.4
Protracted current dysphoria	1.9	2.4
Brief previous dysphoria	11.9	18.7
Protracted previous dysphoria	21.5	28.6
Never dysphoric	61.9	47.8

Table 4. Proportions of participants with and without childhood loss across dysphoria groups

	No loss (<i>N</i> = 479) (%)	Loss (<i>N</i> = 78) (%)
Brief current dysphoria	2.5	2.6
Protracted current dysphoria	1.9	3.9
Brief previous dysphoria	25.9	21.8
Protracted previous dysphoria	14.0	25.6
Never dysphoric	55.7	46.2

nearly one-third (30.3 per cent) of the males who had experienced early childhood loss met criteria for PPD, fewer than one-tenth (9.3 per cent) of those without loss had PPD.

In contrast to the relation between childhood loss and lifetime episodes of dysphoria, parental loss was not significantly associated with the severity of current depressive symptomatology ($t(555) = 1.13$), or with the severity of worst lifetime episode of depression ($t(547) = 1.42$, both $p > .05$). Finally, the main effect of parental loss was not significantly associated with extraversion-introversion ($t(555) = 1.10$), defensiveness ($t(555) = 1.53$), or neuroticism ($t(555) < 1$, all $ps > .10$). There was, however, a significant interaction of parental loss and gender in predicting neuroticism ($F(1,550) = 5.79$, $p < .05$). For males, there was a marginally significant trend for parental loss to be associated with higher levels of neuroticism ($t(258) = 1.71$, $p < .09$), whereas for females, loss was not associated with neuroticism ($t(295) = 1.33$, $p > .10$).

Personality. Participants in the five groups differed significantly on levels of neuroticism ($F(4,552) = 38.03$, $p < .001$), and gender did not moderate this effect. As can be seen in Table 1, follow-up Tukey tests revealed that individuals with PCD reported significantly greater neuroticism than did individuals in each of the other groups (all $ps < .001$), except the BCD group. Most importantly, ND participants had significantly lower levels of neuroticism than did each of the other groups (all $ps < .001$). Finally, the PBD and PPD participants did not differ significantly from each

other with respect to neuroticism. Because PPD and PBD individuals also reported greater current depressive symptomatology than did ND individuals, a one-way analysis of covariance (ANCOVA) was conducted on neuroticism with these three groups, using current depressive symptomatology as a covariate. This analysis also yielded a significant effect ($F(2,527) = 9.84, p < .001$); ND participants exhibited lower levels of neuroticism than did both PPD ($p < .05$) and PBD ($p < .001$) participants.

In contrast to the findings for neuroticism, there were no significant main effects of dysphoria status on extraversion-introversion ($F(4,552) < 1$), or defensiveness ($F(4,552) < 1$). However, there was a significant interaction between the dysphoria status and gender in predicting extraversion ($F(4,547) = 3.54, p < .01$). For males, the five dysphoria groups differed in terms of levels of extraversion ($F(4,255) = 3.16, p < .05$). More specifically, Tukey tests revealed that PCD males were more introverted than were PBD, PPD and ND males (all $ps < .05$). For females, there were no significant differences in extraversion across dysphoria groups ($F(4,292) = 1.25, p > .10$).

Mediation analyses

As we discussed earlier, we hypothesized that the impact of gender and early childhood loss on lifetime experiences of dysphoria would be mediated by neuroticism. This model was tested with path analysis (Cohen & Cohen, 1983, pp. 352-378), using two multiple regression analyses. In the first analysis, neuroticism was regressed on gender and childhood loss. In the second analysis, lifetime experiences of dysphoria was regressed on gender, childhood loss and neuroticism. Currently dysphoric individuals were excluded from the analyses because neuroticism and current symptomatology are likely confounded (Larsen, 1992). Current depressive symptoms were controlled in each of these analyses for this same reason. For the purpose of these analyses, never dysphoric individuals were coded '1' on lifetime episodes of dysphoria, BPD individuals were coded '2', and PPD persons were coded '3'. Gender was dummy coded, with males equal to '1' and females equal to '2'. Loss also was dummy coded: individuals without childhood loss were coded '1' and those with loss were coded '2'.

The results of these analyses indicated that, whereas gender made a significant contribution to the prediction of neuroticism ($t(527) = 5.46, p < .001, \beta = .194$), loss during childhood did not ($t(527) < 1, \beta = -.011$). With respect to lifetime episodes of dysphoria, childhood loss ($t(526) = 2.13, p < .05, \beta = .081$) and neuroticism ($t(526) = 3.14, p < .01, \beta = .147$), each made significant independent contributions, but gender did not ($t(526) = 1.39, p > .10, \beta = .055$). Figure 1 displays this path model with standardized beta coefficients for statistically significant paths. Although gender failed to make a direct contribution to the prediction of dysphoric episodes, female gender was associated with higher levels of neuroticism which, in turn, predicted lifetime episodes of dysphoria. In contrast, childhood loss was unassociated with neuroticism and, instead, made a direct contribution to lifetime episodes of dysphoria.

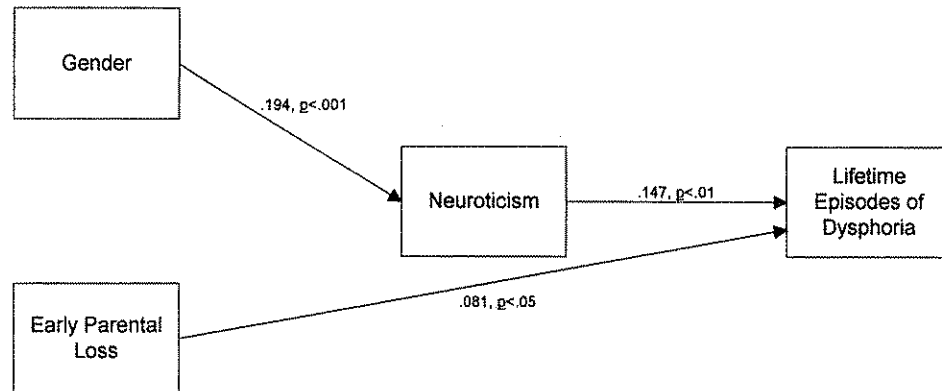


Figure 1. Model of relations among gender, early childhood loss, neuroticism and lifetime episodes of dysphoria after statistically controlling for severity of current depressive symptomatology.

Consistent with earlier analyses indicating gender differences in sensitivity to loss, males and females showed different patterns in these regression analyses when data for each gender were analysed separately. In the case of males, both early parental loss ($t(224) = 2.56, p < .05, \beta = .145$) and neuroticism ($t(244) = 2.06, p < .05, \beta = .143$) significantly contributed to lifetime episodes of dysphoria. This finding again indicates that, for males, neuroticism does not mediate the association between early loss and history of dysphoria, and that loss makes a direct contribution. For females, neuroticism contributed to lifetime episodes of dysphoria ($t(279) = 2.26, p < .05, \beta = .139$), but early parental loss did not ($t(279) < 1, \beta = .039$). Because early parental loss also failed to predict lifetime episodes of dysphoria among females in a regression analysis that excluded neuroticism ($t(280) < 1, \beta = .029$), there was no evidence of mediation in females.

Discussion

The current data suggest that risk for lifetime episodes of dysphoria is associated with being female, experiencing parental divorce or death during childhood and neuroticism. College-age females were 1.6 times more likely than males to experience an episode of protracted dysphoria in their lives, and 1.3 times more likely to experience an episode of brief dysphoria. Similarly, they reported more severe depressive symptoms, both at the time of testing as well as during their worst lifetime depressions. Participants who experienced a parental loss involving death or divorce before the age of 11 were 1.8 times as likely as their 'no loss' counterparts to have had an episode of protracted dysphoria in their lives.¹ Consistent with the results of some studies that have examined the impact of parental divorce (e.g. Hetherington, Cox

¹We also asked participants to briefly describe what they perceived as the cause of their worst lifetime episode of depression. In only a few cases did these descriptions refer to parental death or divorce, suggesting that these episodes of dysphoria were not simply bereavement reactions.

& Cox, 1979; Wallerstein & Kelly, 1980), this effect was limited to males (but see also Rodgers, 1994). Males who experienced parental loss were 3.3 times more likely than males without loss to have experienced an episode of protracted dysphoria. In contrast, females with early childhood loss were no more likely to have a history of dysphoric episodes than were females without loss. Finally, individuals with lifetime episodes of dysphoria obtained higher scores on neuroticism than did participants who reported never having met symptom criteria for major depression (never dysphoric persons). These dysphoria prone individuals consisted of persons who were currently dysphoric, as well as those who had previous episodes of either brief or protracted dysphoria. Importantly, this latter finding remained significant even after current levels of depressive symptoms were statistically controlled. Thus, neuroticism appears to be a personality feature with trait-like characteristics in dysphoria prone persons: it is present not only during, but also following, episodes of dysphoria, and cannot be attributed simply to current symptomatology. In contrast, extraversion-introversion was unrelated to lifetime episodes of dysphoria in this sample of college students.

Although gender differences in vulnerability to depression are robustly established, there is little consensus concerning the mechanism by which being female confers risk. Importantly, our data suggest the operation of one potential mechanism, namely personality differences between males and females. More specifically, the relation between gender and lifetime episodes of dysphoria obtained in the current data was mediated entirely by neuroticism. These results suggest that gender contributes to vulnerability to dysphoria and depression through its association with neuroticism. That is, the gender difference in depression is due, at least in part, to women generally having higher levels of neuroticism than do men. Indeed, women are more likely to develop episodes of dysphoria *only* if they experience heightened neuroticism. In fact, women who report lower neuroticism are less vulnerable to dysphoria than are men who report higher neuroticism. On the other hand, in the current study women were generally less sensitive than men to the effects of early childhood loss (cf. Rodgers, 1994). College-age men who experienced loss tended to report higher levels of neuroticism and were more likely to have a lifetime history of protracted dysphoria than were men without early childhood loss. In contrast, college-age women with loss were at no greater risk than those without loss. These data suggest that boys are less likely than girls to successfully negotiate the challenges and stress associated with early parental loss through death and divorce. Unfortunately, because we did not inquire about which parent was lost, our data cannot address the possibility that maternal versus parental loss differentially affects girls and boys.

In contrast to the relation between gender and dysphoria, the association between early childhood loss and lifetime history of dysphoria was not mediated by neuroticism. Although individuals who had experienced such a loss (particularly males) were at heightened risk for developing protracted episodes of dysphoria, they did not report higher levels of neuroticism. This finding suggests that a different etiological pathway to dysphoria is involved. Future research might examine other potential psychological and environmental mediators, such as parental care (Harris *et al.*, 1990a, b) and working models of attachment relationships (Carnelley *et al.*, 1994;

Roberts *et al.*, 1996b). We also should note that although loss as a main effect failed to predict neuroticism, there was a significant interaction of loss and gender, such that males who experienced loss showed a trend towards higher levels of neuroticism, whereas this was not the case with females. However, additional analyses indicated that neuroticism also failed to mediate the association between loss and lifetime episodes of dysphoria among males.

In addition to basic findings concerning lifetime risk for episodes of dysphoria, the current study has important practical implications with respect to how normal control groups are defined and selected in depression research. As others have noted (e.g. Clark *et al.*, 1994), normative samples are often problematic because they include persons with major depression or a history of major depression. It is obvious that researchers need to exclude such depression prone individuals from their normal control groups. However, our research suggests that, even following such a selection process, normal control groups remain heterogeneous. In particular, a large percentage of these never depressed participants are likely to have met DSM-III-R symptom criteria for major depression without ever having met the two-week duration criteria. The present findings, as well as those from another recent project in our laboratory (Roberts, Gilboa & Gotlib, 1997), suggest that these individuals with brief episodes can be differentiated from pure, never dysphoric, individuals on theoretically important psychological characteristics, such as neuroticism and ruminative response style. Perhaps the most conservative recommendation would be to treat these individuals as a separate group, as we have done in the current study.

Although the current study advances our understanding of vulnerability to lifetime episodes of dysphoria, it also has several limitations. First, our evaluation of childhood loss was limited to the presence of parental death or divorce before the age of 11 years. Due to time constraints during participant assessment, no information was available concerning which parent died or, in the case of divorce, which parent was awarded custody. Similarly, we did not evaluate the quality of relationships with parents, parental conflict, or parenting style, all of which are considered to be important mediators between early childhood loss and subsequent psychological outcomes, such as depression (e.g. Emery, 1982; Franklin *et al.*, 1990; Parker, 1992). Second, our assessment of history of episodes of dysphoria was limited to self-reports in a relatively healthy sample of college students. Future research would benefit from evaluations of depression based on structured interviews with more severe, clinical, samples. Third, further studies are also required to delineate the processes and mechanisms by which neuroticism affects vulnerability to dysphoria and depression. For example, recent studies indicate that negative attentional orienting biases are associated with neuroticism (Derryberry, 1987; Derryberry & Reed, 1994; Reed & Derryberry, 1995), whereas Watson & Pennebaker (1989) suggested that individuals with high negative affectivity or neuroticism are hypervigilant to bodily symptoms and pain. Our own research (Nolan, Roberts & Gotlib, in press; Roberts *et al.*, 1997) suggests that neuroticism is associated with a ruminative response style, in which individuals repetitively focus on their depressive moods and symptoms, leading to more persistent and severe episodes of dysphoria. Together, these studies suggest that neuroticism increases risk for dysphoria (and perhaps depres-

sion) by negatively biasing cognitive processes, including attentional focus and memory (Martin, 1985). It remains for future research to examine this formulation more explicitly.

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